## **AMENDMENTS TO THE CLAIMS**

Please enter the following amendments to the claims without prejudice, without admission, without surrender of subject matter, and without any intention of creating any estoppel as to equivalents as follows:

- 1. (Currently amended) A transgenic mouse model <u>showing hypomyelinosis of the</u> thalamus <u>of oligodendrocyte developmental disorders</u>-wherein the transgenic mouse comprises a <u>homozygous</u> disruption in chromosomal DAP12 (DNAX Activation Protein 12) gene function, and wherein the <u>transgenic mouse shows hypomyelinosis of the thalamus homozygous</u> <u>disruption includes the promoter region and exons 1, 2, and 3</u>.
  - 2. (Canceled)
- 3. (Currently amended) The transgenic mouse model of claim 1, wherein the homozygous disruption in DAP12 can be pheonotypically exhibited as oligodendrocyte developmental disorder is a myelinogenesis developmental disorder or a neuropsychiatric disorder.
- 4.(Currently amended) The transgenic mouse model of claim 3, wherein the neuropsychiatric disorder is selected from the group consisting of Nasu-Hakola disease, dementia, schizophrenia, schizotypal personality disorders, obsessive-compulsive disorders, Huntington's disease or Tourette's syndrome.
- 5. (Currently amended) The transgenic mouse model of claim 3, wherein the neuropsychiatric disorder is Nasu-Hakola disease or dementia.
  - 6-18. (Canceled)
- 19. (Previously presented) The transgenic mouse model of claim 1, wherein the expression of myelin basic protein in the brain is weak in regions where DAP12 is strongly expressed in wild-type mice.
- 20. (Previously presented) The transgenic mouse model of claim 1, wherein the transgenic mouse exhibits an impairment in sensorimotor gating as compared to wild-type mice.